Laboratory Estimates of Heritabilities and Genetic Correlations in Nature

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ABSTRACT

A lower bound on heritability in a natural environment can be determined from the regression of offspring raised in the laboratory on parents raised in nature. An estimate of additive genetic variance in the laboratory is also required. The estimated lower bounds on heritabilities can sometimes be used to demonstrate a significant genetic correlation between two traits in nature, if their genetic and phenotypic correlations in nature have the same sign, and if sample sizes are large, and heritabilities and phenotypic and genetic correlations are high.

A recurring problem in evolutionary biology is the relationship of parameter estimates derived from laboratory or greenhouse studies to the evolutionarily relevant parameter values in nature (PROUT 1958; MITCHELL-OLDS and RUTLEDGE 1986). With some exceptions (*e.g.*, BOAG and GRANT 1978; SMITH and DHONDT 1980; VAN NOORDWIJK, VAN BALEN and SCHARLOO 1980; SHAW 1986; MITCHELL-OLDS 1986; GIBBS 1988), heritabilities and other genetic parameters are estimated under controlled laboratory conditions designed to minimize environmental variation. The laboratory may also differ from natural environments in ways that alter the relative values of different genotypes, causing genotype-by-environment interaction that can bias estimates of heritabilities and genetic correlations (FALCONER 1952; GUPTA and LEWONTIN 1982; VIA 1984; SERVICE and ROSE 1985). These parameters may also differ among natural populations of a single species (LOFSVOLD 1986; DINGLE, EVANS and PALMER 1988; COHAN and HOFFMANN 1989); and, within a particular population, they can be affected by spatially and temporally varying conditions (SERVICE 1984; SERVICE and ROSE 1985; GILLESPIE and TURELLI 1989; PROUT and BARKER, 1989). To gather more information on the variation of these parameters, it is important to develop estimation techniques that circumvent the difficulties of estimating genetic parameters in nature. In the following, we will treat the parameter values in nature and in the laboratory as constants, but our method can be used to test this assumption.

In an appendix to COYNE and BEECHAM (1987), LANDE showed that bounds could sometimes be set on heritabilities in nature by regressing offspring raised in the laboratory on their parents raised in the natural environment. This bound, however, only applies when the estimated additive genetic variance in the laboratory is less than the estimated covariance between offspring in the laboratory and midparent in nature. Here we extend LANDE's approach to show how a lower bound on the heritability in nature can often be estimated even when LANDE's condition is not met. Also, this lower bound can sometimes be used to obtain information about the genetic correlation in nature. Throughout our analysis, we assume that the distributions of breeding values for the traits of interest are Gaussian, that the trait values are scaled so that males and females have the same means and variances, and that maternal effects, epistasis and selection are negligible.

HERITABILITY

In LANDE's notation, the regression of laboratory offspring on midparent from nature is

$$\beta_{(O_L \cdot P_N)} = \frac{\gamma \sigma_{AL} \sigma_{AN}}{\sigma^2_{PN}} \tag{1}$$

where γ is the additive genetic correlation between the trait in nature and the same trait in the laboratory (FALCONER 1952), σ^2_{AL} and σ^2_{AN} are the additive genetic variances in the laboratory and in nature, and σ^2_{PN} is the phenotypic variance in nature. To obtain a lower bound for the heritability in nature, $h^2_N = \sigma^2_{AN}/\sigma^2_{PN}$, note that

$$\beta^2_{(O_L \cdot P_N)} \left(\frac{\sigma^2_{PN}}{\sigma^2_{AL}} \right) = \gamma^2 h^2_N \leq h^2_N. \tag{2}$$

An estimate, V_{PN}, of σ^2_{PN} can be obtained from the phenotypic variance of the parents from nature. (This may differ from an estimate derived from a random sample in nature, if some phenotypic classes do not mate or are infertile.) An estimate V_{AL} of σ^2_{AL} can be obtained from parent-offspring regression when both are raised in the laboratory, or from the variance

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among half-sib families in the laboratory (FALCONER 1981, Ch. 10). If b is an empirical estimate of β, then

$$b^2_{(O_L \cdot P_N)}\left(\frac{V_{PN}}{V_{AL}}\right) \tag{3}$$

is an estimate of $\gamma^2 h_N^2$, which is a lower bound for both h_N^2 and γ^2. If, as is often true, only mothers can be identified in nature, the squared regression coefficient of offspring on parent should be multiplied by 4 because the regression on one parent is half that on the midparent (FALCONER 1981, p. 151). If the estimate V_{AL} is inflated by dominance, common environment, or maternal effects, *e.g.*, if V_{AL} is derived from full-sib covariances in the laboratory, the lower bound derived from (3) becomes more conservative, unless the estimate of $\beta_{(O_L \cdot P_N)}$ is also affected, as by maternal effects. Estimate (3) is available whenever V_{AL} is nonzero. The bound for h_N^2 is best when γ^2 is large and useless when γ^2 is zero, but the magnitude of γ^2 will be unknown unless covariances among relatives in nature are available. Note that if $h_N^2 = 0$ then $\sigma_{AN}^2 = 0$ and γ is undefined, so an estimate of $\beta_{(O_L \cdot P_N)}$ that differs significantly from zero, regardless of sign, is sufficient evidence for $h_N^2 > 0$, but does not reveal the magnitude of h_N^2.

$\gamma^2 h_N^2$ can be viewed as an approximation to h_N^2 that is biased when $|\gamma| \neq 1$. Another simple approximation of heritability in nature is the ratio of the additive genetic variance in the laboratory to the phenotypic variance in nature, $h_V^2 = \sigma_{AL}^2/\sigma_{PN}^2$. This approximation avoids bias arising from $|\gamma| \neq 1$, but not that arising from differences between σ_{AL}^2 and σ_{AN}^2. When the offspring-parent regression $\beta_{(O_L \cdot P_N)}$ is used as an approximation for h_N^2, both kinds of bias are present. Unfortunately, these two biases cannot both be readily eliminated by any of these approximations. The relationships between these three approximations of heritability can be understood by considering their ratios:

$$k = \frac{h_V^2}{\beta_{(O_L \cdot P_N)}} = \frac{\beta_{(O_L \cdot P_N)}}{\gamma^2 h_N^2} = \left(\frac{\sigma_{AL}}{\sigma_{AN}}\right)\frac{1}{\gamma}. \tag{4}$$

If $|k| < 1$, then because $|\gamma| < 1$, $\sigma_{AL}^2 < \sigma_{AN}^2$ showing that additive genetic variance is larger in nature than in the laboratory. This is LANDE's criterion in COYNE and BEECHAM (1987). When this relationship holds, $h_V^2 < |\beta_{(O_L \cdot P_N)}| < \gamma^2 h_N^2$, so that the lower bound $\gamma^2 h_N^2$ is larger than the other two approximations and these are therefore too small. If $|k| > 1$ then, as LANDE noted, nothing is known about the relative magnitudes of genetic variances in the two environments; but $\gamma^2 h_N^2$ can still be estimated. Also, when $|k| > 1$, then $h_V^2 > |\beta_{(O_L \cdot P_N)}| > \gamma^2 h_N^2$, so that the lower bound $\gamma^2 h_N^2$ is the smallest of the three approximations. One of the other approximations could then be the closest to h_N^2, but the data provide no way of deciding whether or not this is so.

ASSORTATIVE MATING

These estimates can also be used when parents have been mated assortatively, if one is willing to assume that all genetic variance is additive. The slope of the parent-offspring regression is unchanged by assortative mating within a single environment (FALCONER 1981, Ch. 10; for exceptions see GIMELFARB 1985). This is also true across environments. Let $M_{P(N)}$ be the midparent phenotype in nature, $M_{O(L)}$ the expected phenotype of their offspring in the laboratory, $P_{S(N)}$ the sire phenotype, $P_{D(N)}$ the dam phenotype, and r the phenotypic correlation between parents due to assortative mating. Because we are interested in laboratory-reared offspring of parents from nature, we must regress the parents' breeding values for the trait as expressed in the laboratory on their phenotypes in nature. Denoting these sire and dam breeding values by $A_{S(L)}$ and $A_{D(L)}$, the regressions are

$$A_{S(L)} = \beta_{LN} P_{S(N)} + R_S \tag{5a}$$

and

$$A_{D(L)} = \beta_{LN} P_{D(N)} + R_D. \tag{5b}$$

The regression coefficient, β_{LN}, equals $\beta_{(O_L \cdot P_N)}$ from (1). The residuals R_S and R_D are independent of each other and the phenotypic values, and each has variance $\sigma_{AL}^2[1 - (\gamma^2 \sigma_{AN}^2/\sigma_{PN}^2)]$. In this notation,

$$M_{P(N)} = \tfrac{1}{2}(P_{S(N)} + P_{D(N)}) \tag{6}$$

and

$$M_{O(L)} = \text{constant} + \tfrac{1}{2}(A_{S(L)} + A_{D(L)}). \tag{7}$$

Thus,

$$\begin{aligned}\text{Cov}(M_{P(N)}, M_{O(L)}) &= \tfrac{1}{4}(\beta_{LN}\sigma^2_{P_{S(N)}} + \beta_{LN}\sigma^2_{P_{D(N)}} + 2\beta_{LN} r \sigma_{P_{S(N)}}\sigma_{P_{D(N)}}) \\ &= \tfrac{1}{2}\gamma\sigma_{AL}\sigma_{AN}(1 + r),\end{aligned} \tag{8}$$

which, if $L \equiv N$, is $\tfrac{1}{2}\sigma_A^2(1 + r)$. Similarly, the variance of the midparent values is

$$\text{Var}(M_{P(N)}) = \tfrac{1}{2}\sigma_{PN}^2(1 + r). \tag{9}$$

Thus, assortative mating has no effect on the slope of the offspring-midparent regression, either within or across environments, because both the numerator (8) and denominator (9) are multiplied by the same factor, $(1 + r)$. [This need not be true if our assumptions are not met; for example, if regressions are nonlinear (GIMELFARB 1985).]

Sib covariances, however, are affected by assortative mating (FALCONER 1981, Ch. 10). For example, the covariance of full sibs in the lab is the variance of (7), i.e.,

$$\text{Cov}(O_1, O_2) = \tfrac{1}{2}\sigma_{AL}^2(1 + r\gamma^2 h_N^2). \tag{10}$$

If the natural and laboratory environments are the same, so that $\gamma = 1$ and $\sigma_{AL}^2 = h_L^2\,\sigma_{PN}^2$, then (10) is the

basis for the standard estimate of h^2 with assortative mating (FALCONER 1981, p. 164). If only the environmental variances differ between laboratory and nature, (10) alone suffices to estimate h_N^2; without making this assumption, (3) and (10) can be solved together for $\gamma^2 h_N^2$ (see PROUT and BARKER 1989). Equation 10 can also be used to estimate $\gamma^2 h_N^2$ if the experiment is performed for different values of r. For example, this can be done by substituting an estimate of σ_{AL}^2 obtained when $r = 0$ into (10) for a second experiment with $r \neq 0$, and solving for $\gamma^2 h_N^2$. More generally, (10) can be written as the regression equation $y = \beta_0 + \beta_0\beta_1 x + e$, where y is a sample covariance between full sibs, x is the corresponding value of r, $\beta_0 = \frac{1}{2}\sigma_{AL}^2$, $\beta_1 = \gamma^2 h_N^2$, e is a residual, and the equation is nonlinear in the parameters. For experiments having different values of r, nonlinear least squares or similar methods might be used to estimate β_0 and β_1. Each experiment could also provide a separate estimate of the offspring-midparent regression (1), and estimates of $\gamma^2 h_N^2$ derived from these could be compared with those derived from sib covariances (10). One source of discrepancy between these different estimates of $\gamma^2 h_N^2$ is dominance variance, which does not enter into the regression estimate, but does affect sib covariances [(10) assumes no dominance]. If the two kinds of estimates do not differ statistically, data from all of the experiments could be combined in one analysis to estimate a lower bound for h_N^2 (GIANOLA 1979). Alternatively, one may wish to avoid this use of assortative mating if dominance or common environments are suspected of contributing to full-sib covariances. Assortative mating also complicates the analysis of multiple traits and genetic correlations.

These derivations for assortative mating depend critically on the assumption that the correlation r between sire and dam phenotypes is caused by assortment on the particular trait measured, and does not arise indirectly from assortment on any other trait (cf. FALCONER 1981, pp. 161–162). Under this assumption, the resulting correlation between sire and dam breeding values for this trait depends only on its heritability. Effects of assortment with respect to one trait on the breeding values of a second trait, however, depend on both the heritability of the first trait and its genetic correlation with the second trait. For example, (8) through (10) apply whether "L" and "N" are different traits in the same environment, the same trait in different environments, or different traits in different environments, as long as r measures the correlation between sire and dam for trait N, and the phenotype of N was the criterion for assortative mating. If, however, the assortment criterion was the phenotype of some other trait, then (8) and (10) may be invalid, and offspring-midparent regressions can be changed by assortative mating. An extreme example will make this clear. Suppose that the criterion for assortment is a non-heritable trait environmentally correlated with N. The sire and dam phenotypes for N will be correlated because of their environmental correlations with the assortment criterion, so a nonzero r could be calculated between sire and dam phenotypes for N. This r would affect the variance of the midparent values for N as shown in (9). The appropriate r for (8), however, would be zero, because assortment by a non-heritable trait will not affect the breeding values of any trait. In this example, therefore, assortative mating would change the offspring-midparent regression slope, because its denominator (9) would be changed, but its numerator (8) would not. As this example shows, the causal paths that generate r are important, so one cannot simply measure a correlation between sire and dam and insert the resulting r into the formulae. If a correlation is observed between sires and dams that have paired naturally, application of (8) through (10) assumes that the measured trait, and no other, was the criterion of assortment.

GENETIC CORRELATION

Next, consider two traits in nature, X and Y, and their homologous expressions in the laboratory environment, X' and Y'. The lower bounds on the heritabilities h_X^2 and h_Y^2 estimated from (3) can sometimes be combined with an estimate of phenotypic correlation to determine the sign of the additive genetic correlation in nature. Let $e^2 = 1 - h^2$, and let ρ_e be the "environmental" correlation [including nonadditive genetic covariance (FALCONER 1981, p. 282)] between X and Y in nature. As before, additive genetic correlation will be represented by γ, but now with subscripts to indicate the traits and environments involved.

The phenotypic correlation in nature, ρ_p, is a weighted sum of the genetic and environmental correlations:

$$\rho_p = h_X \gamma_{XY} h_Y + e_X \rho_e e_Y. \tag{11}$$

A lower bound for h^2 provides an upper bound for $e^2 = 1 - h^2$, so the estimated lower bounds for h^2 from (3) can be used together with the fact that $|\rho_e| \leq 1$ to bound $e_X \rho_e e_Y$. By setting bounds on $e_X \rho_e e_Y$, we can sometimes show that γ_{XY} has the same sign as ρ_p. The maximum environmental contribution to ρ_p occurs when ρ_e has the same sign as ρ_p and $|\rho_e| = 1$. If

$$|\rho_p| > \sqrt{(1 - h_X^2)(1 - h_Y^2)}, \tag{12}$$

or equivalently,

$$\rho_p^2 > (1 - h_X^2)(1 - h_Y^2), \tag{13}$$

then environmental correlation cannot account for the magnitude of the phenotypic correlation. The

additional correlation must therefore be genetic, and γ_{XY} must therefore have the same sign as ρ_p when (13) is true. The use of estimated lower bounds for h^2 from (3) in (13) will provide a very conservative test. Alternatively, we could set bounds for γ_{XY} by finding the minimum and maximum of

$$\rho_p \pm \frac{\sqrt{(1 - h_X^2)(1 - h_Y^2)}}{h_X h_Y} \qquad (14)$$

for allowable values of h^2, i.e., values of h_x^2 *and* h_Y^2 between unity and the minima estimated from (3). Another lower bound for γ_{XY} could be obtained by using (1) and (3) to estimate bounds for the genetic correlations between traits in different environments, which could be large enough to place a lower limit on the genetic correlation in nature. Unfortunately, unless heritabilities are atypically high, a lower bound obtained by this method would probably be too low to be useful.

Assortative mating will complicate analyses of genetic correlation because only one of the traits X or Y can be used as the assortment criterion. Equations 8 and 10 will therefore not generally apply to the other trait, and $\gamma^2 h_N^2$ cannot be estimated for this second trait without additional information or assumptions. Because of this, assortative mating should probably be avoided if more than one trait is to be analyzed.

An alternative to (13–14) is to ignore differences between environments and apply the usual formula for estimating genetic correlation between traits, assuming that parents and offspring were measured in the same environment (FALCONER 1981). COYNE and BEECHAM (1987, Table 4) estimated genetic correlations in nature as the arithmetic mean of the two reciprocal between-trait offspring-midparent covariances divided by the geometric mean of the within-trait offspring-midparent covariances. This formula estimates $\frac{1}{2}(\sigma_X\sigma_{Y'}\gamma_{XY'} + \sigma_X \cdot \sigma_Y\gamma_{X'Y})/\sqrt{\sigma_X\sigma_{X'}\sigma_Y\sigma_{Y'}\gamma_{XX'}\gamma_{YY'}}$, where σ denotes the additive genetic standard deviation. The usefulness of this estimate of γ_{XY} depends critically on the magnitude of genotype-environment interaction, because the quantity estimated contains neither the desired between-trait genetic correlation in nature, γ_{XY}, nor the between-trait genetic correlation in the laboratory, $\gamma_{X'Y'}$.

EXAMPLES

We have applied these methods to data from COYNE and BEECHAM (1987) on wing length and bristle count (sum of fourth and fifth abdominal segments) for *Drosophila melanogaster*. Data from two experiments were kindly provided by J. COYNE. In the first (experiment 1A, 142 families, 2 offspring of each sex per family), offspring of wild-caught parents were raised in the laboratory. In the second experiment (1B, 159 families, 2 offspring of each sex per family), parents and their offspring were both raised in the laboratory. Before computing estimates, Wright's modification of the logarithmic transformation (FALCONER 1981, p. 266) was applied to equalize within-environment phenotypic variances between the sexes. This transformation uses the ratio of the intercept and slope from the regression of standard deviation on mean to compute a value to be added to observations before log-transforming. Very nearly the same ratio was obtained for the two environments (−0.6894 and −0.6776). The average of these (−0.6835) was added to both the natural and laboratory trait values, and the natural logs of these sums were the traits analyzed. This transformation resulted in nearly equal phenotypic variances for the two sexes within environments. A square-root transformation was used to decrease differences in variance for the bristle counts (SOKAL and ROHLF 1981).

$b^2_{(O_L \cdot P_N)}$ was computed as the squared sample regression of family means in the laboratory on midparent in nature from experiment 1A. V_{PN} was estimated as the variance of the midparent in experiment 1A multiplied by $2/(1 + r)$, where r was the correlation between dam and sire. V_{AL} was obtained from experiment 1B by multiplying the covariance between offspring mean and midparent by $2/(1 + r)$, where r was again the phenotypic correlation between sire and dam. We used r in these calculations to avoid computing separate phenotypic variances for sires and dams. This involves no approximation. In general, though, our analyses of heritability and genetic correlation were only approximate, because we assumed that the trait being analyzed was also the criterion of assortment. In these experiments, however, assortative mating was by general size instead of the measured traits (COYNE and BEECHAM 1987, p. 729). Because the correlations were low, this approximation may not have greatly affected the results.

An estimate of the distribution of (3) was obtained by bootstrapping (EFRON 1982; RIPLEY 1987). Each bootstrap sample was drawn from the data by sampling (with replacement) 142 times from the 142 families in experiment 1A, and 159 times from the 159 families in experiment 1B, assuming independence of the two experiments. The estimate (3) was computed directly from the data and also from each of 1000 bootstrap samples. The bias-corrected percentile method (EFRON 1982, p. 82) was used to obtain approximate confidence limits from the bootstrap distribution, and the closest bootstrap value to the bias-corrected fiftieth percentile was taken as the bootstrap estimate of the parameter (column 2 in Tables 1 and 2). A problem that can arise in the bootstrapping (and also in direct estimation) is that the V_{AL} estimate can sometimes be negative. This happens when the laboratory parent-offspring regression slope for the sample is negative. Also, if this slope is very close to zero, extreme estimates of $\gamma^2 h_N^2$ that are well outside the

TABLE 1

Heritability analysis of *D. melanogaster* wing lengths and bristle counts

Trait	Direct estimate of $\gamma^2 h_N^2$	Bootstrap $\gamma^2 h_N^2$ (bias-corrected percentile method)[a]						
		Bootstrap estimate	Upper and lower 95% limits		Lower (one-tailed) 95% limit	h_V^2[b]	$b_{(OL \cdot PN)}$	k[c]
Males								
Wing length	0.1253	0.1188	0.0106	0.3790	0.0206	0.260	0.180	1.44
Bristle count	0.6118	0.5800	0.1135	2.1984	0.1660	0.323	0.444	0.73
Females								
Wing length	0.2487	0.2492	0.0375	0.6982	0.0605	0.248	0.248	1.00
Bristle count	0.3596	0.3524	0.0468	0.1444	0.0705	0.564	0.451	1.25

[a] Bootstrap analyses are each based on 1000 bootstrap samples.
[b] $h_V^2 = \sigma_{AL}^2/\sigma_{PN}^2$.
[c] $k = \sigma_{AL}/(\gamma\sigma_{AN})$.

TABLE 2

Bootstrap analyses of genetic correlation

Trait	Direct estimate	Bias-corrected percentile method[a]			
		Bootstrap estimate	Upper and lower 95% limits		Lower (one-tailed) 95% limit
Male wing and bristle					
r_p	0.3128	0.3098	0.2060	0.4011	0.2252
Wing: $\gamma^2 h_N^2$	0.1253	0.1199	0.0124	0.3804	0.0184
Bristle: $\gamma^2 h_N^2$	0.6118	0.6241	0.1311	2.3380	0.1939
$r_p^2 - (1 - h_X^2)(1 - h_Y^2)$	−0.2417	−0.2539	−0.6858	1.0776	−0.6359
Female wing and bristle					
r_p	0.3128	0.3096	0.2113	0.4018	0.2243
Wing: $\gamma^2 h_N^2$	0.2487	0.2543	0.0542	0.7003	0.0718
Bristle: $\gamma^2 h_N^2$	0.3596	0.3739	0.0390	1.2681	0.0852
$r_p^2 - (1 - h_X^2)(1 - h_Y^2)$	−0.3833	−0.4119	−0.7676	0.0970	−0.7403
4th- and 5th-segment bristle counts (sexes pooled)					
r_p	0.4900	0.4841	0.3881	0.5742	0.4042
Seg. 4: $\gamma^2 h_N^2$	0.4491	0.4207	0.0196	2.1767	0.0484
Seg. 5: $\gamma^2 h_N^2$	0.3551	0.3487	0.0579	1.0148	0.0886
$r_p^2 - (1 - h_X^2)(1 - h_Y^2)$	−0.1152	−0.1658	−0.7019	0.6184	−0.6189
Simulated data for 150 families					
r_p	0.9028	0.9024	0.8839	0.9183	0.8849
Trait X: $\gamma^2 h_N^2$	0.3611	0.3501	0.1248	0.7793	0.1487
Trait Y: $\gamma^2 h_N^2$	0.3481	0.3457	0.1382	0.7621	0.1624
$r_p^2 - (1 - h_X^2)(1 - h_Y^2)$	0.3985	0.3775	0.0698	0.7214	0.1086

[a] Bootstrap analyses are each based on 1000 bootstrap samples.

zero-to-one range of possible parameter values can occur. For male bristle count, we obtained 4 negative bootstrap estimates of V_{AL} out of 1000, and a few extreme bootstrap estimates of $\gamma^2 h_N^2$ indicated that the corresponding V_{AL} estimates must have been very close to zero. Here, negative estimates of V_{AL} corresponded to the extremes of sampling variance from the offspring-midparent distribution, and contributed to the extreme tail of the bootstrapped distribution.

Results are shown in Table 1 and Figure 1. All of the bootstrap estimates of $\gamma^2 h_N^2$, the lower bound for heritability in nature, are significantly greater than zero at the 5% level. All of our examples yielded estimates of $\beta_{(O_L \cdot P_N)}$ corresponding to positive values of γ. Table 1 also shows estimates of $h_V^2 = \sigma_{AL}^2/\sigma_{PN}^2$, $\beta_{(O_L \cdot P_N)}$, and the ratio $k = \sigma_{AL}/(\gamma\sigma_{AN})$. k was less than one for bristle count in males and slightly less than one for wing length in females. According to LANDE's criterion (COYNE and BEECHAM 1987), $\beta_{(O_L \cdot P_N)}$ is therefore a lower bound for h_N^2 only for bristle count in males and wing length in females. For these cases, with $k < 1$, our lower bound $\gamma^2 h_N^2$ is higher than the other two approximations, as it must be (from 4), although for female wing length the three approximations are within rounding error of each other. Comparison of our results with those of COYNE and BEECHAM (1987) is complicated by a transposition of numbers in the appendix of that paper (0.000977 and 0.00157 were interchanged), and a misprint in their Table 4 (J. COYNE, personal communication). The values of k calculated from their table 4, after correcting the misprint (0.00120 should have been 0.00157), are 1.61 for wing length and 1.03 for bristle count. Their results will also differ from ours because

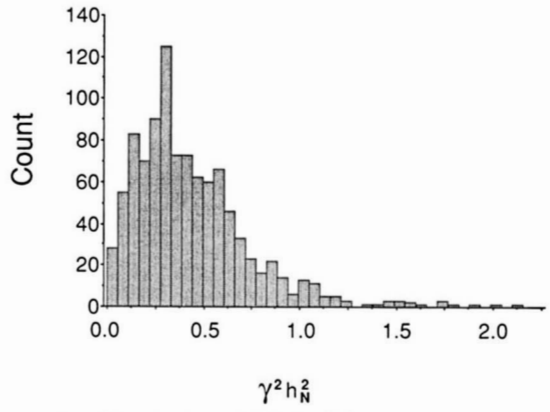

FIGURE 1.—Distribution of 1000 $\gamma^2 h_N^2$ estimates for female *D. melanogaster* bristle counts, each calculated from a separate bootstrap sample.

we analyzed sexes separately and used different transformations. Our estimated lower bounds cannot prove that a trait is less heritable in nature than in the laboratory, so we provide no support for COYNE and BEECHAM's (1987) conclusion that this is so for wing length. Their general result, however, that these traits are heritable in nature, is supported by our analyses.

For comparison with the bootstrap results, we also obtained approximate standard errors for some of our $\gamma^2 h_N^2$ estimates by the delta method (KENDALL and STUART 1969, p. 231). We subtracted twice the approximate standard error from the direct estimate of $\gamma^2 h_N^2$ to obtain approximate lower confidence limits. For the cases examined, these were negative (*e.g.*, −0.384 for bristle count in males), and well below the bootstrap estimates, perhaps because the strong right-skew of the sample statistic, as indicated by the bootstrap results (Figure 1), requires asymmetrical confidence intervals.

We also used (13) to examine correlations between wing length and bristle count separately for males and females, as well as the correlation between the fourth- and fifth-segment bristle counts for pooled data. (Sex-specific offspring data were not available for bristle counts for the separate segments.) To do this, we bootstrapped an estimate of $\rho_p^2 - (1 - h_X^2)(1 - h_Y^2)$ to test against the one-tailed alternative hypothesis that this quantity was greater than zero. $(1 - h_X^2)$ and $(1 - h_Y^2)$ were estimated by subtracting from 1 the $\gamma^2 h_X^2$ and $\gamma^2 h_Y^2$ estimates obtained as above, but here both traits were bootstrapped at the same time, so that the individual bootstrap estimates for the two traits were calculated from the same bootstrap samples of families. Also, a bootstrap estimate of ρ_p^2 was obtained at the same time, from the same bootstrap sample of families. For a given bootstrap sample, the correlation between X and Y was computed separately for sires and dams in nature, each correlation was transformed to Fisher's z (SOKAL and ROHLF 1981, p. 583), and the average of these two z-values was back-transformed to r. The square of this r was the estimate of ρ_p^2. These procedures yielded an estimate of $\rho_p^2 - (1 - h_X^2)(1 - h_Y^2)$ for each bootstrap sample. If the fifth bias-corrected percentile of the bootstrap estimates exceeded zero, we would conclude that the genetic correlation in nature had the same sign as ρ_p. These analyses were approximate, because we have assumed that each trait was the criterion for assortative mating.

Results of these analyses are shown in table 2. None of the bootstrap estimates of $\rho_p^2 - (1 - h_X^2)(1 - h_Y^2)$ were greater than zero, so we could not conclude that the genetic correlation in nature was positive for these traits. In these correlation examples, however, either the heritability estimate, or the phenotypic correlation, or both, were relatively low. Given the very conservative nature of this test, the lack of statistical significance is not surprising. Cases in which both heritabilities and correlations are higher might yield statistically significant results.

To demonstrate this, we generated artificial data for 150 families corresponding to the design of experiment 1A, and 150 corresponding to 1B. We assumed heritabilities of 0.5 in nature and 0.56 in the laboratory, $\gamma_{XY} = \gamma_{X'Y'} = \gamma_{XX'} = \gamma_{YY'} = 0.9$, environmental correlations of 0.9 in nature and 0.86 in the laboratory, and that 5 offspring were measured per family. Six artificial data values per family (two traits for sire, dam, and mean of 5 offspring) were generated from a multivariate normal distribution with covariance determined by the hypothetical genetic parameters assuming no intentional assortative mating. Means, variances, correlations, and parent-offspring regressions from the artificial data were checked against the hypothetical parametric values. The simulated data were then analyzed in the same way as the fly data. All of the results (Table 2) were significant, and only one of the 1000 bootstrap estimates of $\rho_p^2 - (1 - h_X^2)(1 - h_Y^2)$ was less than zero. In another analysis of only 100 simulated families, the correlation test was not significant. The results show that although the test is not very powerful, it is sometimes possible to make a reliable statement about the sign of the genetic correlation in nature if the heritabilities and correlations are high enough, and if enough families are measured.

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LITERATURE CITED

BOAG, P. T., and P. R. GRANT, 1978 Heritability of external morphology in Darwin *finches*. *Nature* **274:** 793–794.

COHAN, F. M., and A. A. HOFFMANN, 1989 Uniform selection as a diversifying force in evolution: evidence from Drosophila. Am. Nat. (in press).

COYNE, J. A., and E. BEECHAM, 1987 Heritability of two morphological characters within and among natural populations of *Drosophila melanogaster*. Genetics **117:** 727–737.

DINGLE, H., K. E. EVANS and J. O. PALMER, 1988 Responses to selection among life-history traits in a nonmigratory population of milkweed bugs (*Oncopeltis fasciatius*). Evolution **42:** 79–92.

EFRON, B., 1982 *The Jackknife, the Bootstrap, and Other Resampling Plans*. S.I.A.M., Philadelphia.

FALCONER, D. S., 1952 The problem of environment and selection. Am. Nat. **86:** 293–298.

FALCONER, D. S., 1981 *Introduction to Quantitative Genetics*, Ed. 2. Longman, London.

GIANOLA, D., 1979 Estimation of genetic covariance from joint offspring-parent and sib-sib statistics. Genetics **93:** 1039–1049.

GIBBS, H. L., 1988 Heritability and selection on clutch size in Darwin's medium ground finches (*Geospiza fortis*). Evolution **42:** 750–762.

GILLESPIE, J. H., and M. TURELLI, 1989 Genotype-environment interactions and the maintenance of polygenic variation. Genetics **121:** 129–138.

GIMELFARB, A., 1985 Is offspring-midparent regression affected by assortative mating of parents? Genet. Res. **47:** 71–75.

GUPTA, A. P., and R. C. LEWONTIN, 1982 A study of reaction norms in natural populations of *Drosophila pseudoobscura*. Evolution **36:** 934–948.

KENDALL, M. G., and A. STUART, 1969 *The Advanced Theory of Statistics*, Vol. 1, Ed. 3. Hafner, New York.

LOFSVOLD, D., 1986 Quantitative genetics of morphological differentiation in *Peromyscus*. I. Tests of the homogeneity of genetic covariance structure among species and subspecies. Evolution **40:** 559–573.

MITCHELL-OLDS, T., 1986 Quantitative genetics of survival and growth in *Impatiens capensis*. Evolution **40:** 107–116.

MITCHELL-OLDS, T., and J. J. RUTLEDGE, 1986 Quantitative genetics in natural plant populations: a review of the theory. Am. Nat. **127:** 379–402.

PROUT, T., 1958 A possible difference in genetic variance between wild and laboratory population. Drosophila Inform. Serv. **32:** 148–149.

PROUT, T., and J. S. F. BARKER, 1989 Ecological aspects of the heritability of body size in *Drosophila buzzatii*. Genetics **123:** 803–813.

RIPLEY, B. D., 1987 *Stochastic Simulation*. Wiley, New York.

SERVICE, P. M., 1984 Genotypic interactions in an aphid-host plant relationship: *Uroleucon rudbeckiae* and *Rudbeckia laciniata*. Oecologia **61:** 271–276.

SERVICE, P. M., and M. R. ROSE, 1985 Genetic covariation among life-history components; the effect of novel environments. Evolution **39:** 943–945.

SHAW, R. G., 1986 Response to density in a natural population of the perennial herb *Salvia lyrata*: variation among families. Evolution **40:** 492–505.

SMITH, J. N., and A. A. DHONDT, 1980 Experimental confirmation of heritable morphological variation in a natural population of song sparrows. Evolution **34:** 1155–1158.

SOKAL, R. R., and F. J. ROHLF, 1981 *Biometry*, Ed. 2. Freeman, San Francisco.

VAN NOORDWIJK, A. J., J. H. VAN BALEN and W. SCHARLOO, 1980 Heritability of ecologically important traits in the great tit. Ardea **68:** 193–203.

VIA, S., 1984 The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. Evolution **38:** 896–905.

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